Case Report

Post-transplant idiopathic non-lupus full-house nephropathy – A case report

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ABSTRACT

We report a case of Idiopathic non-lupus full-house nephropathy (NLFHN) in a 39-year-old male who had a full-house pattern of immunofluorescence study without overt systemic lupus erythematosus after a follow-up of more than 2 years. The incidence of detection of cases of NLFHN is increasing in native kidney biopsy and is critical to report as they have poor clinical outcomes. To the best of our knowledge, it is the first case of post-transplant renal biopsy and needs to be reported to plan the treatment protocol for such transplant patients.

Key words: Electron microscopic study, Full-house nephropathy, Histopathology, Kidney transplant recipient

Full-house pattern of immunofluorescence on kidney biopsy in a patient with any morphological pattern on renal biopsy first suggests the possibility of lupus nephritis which is confirmed by correlating with clinical and serological evidence of systemic lupus erythematosus (SLE) [1,2]. However, in patients with full-house nephropathy and any morphological pattern on renal biopsy without clinical or serological evidence of SLE is termed as non-lupus full-house nephropathy (NLFHN). It is critical to report these cases as they usually have poor clinical outcomes and they differ from SLE cases and lupus nephritis [3,4].

CASE REPORT

A 39-year-old hypertensive male (since 2 years) presented to the nephrologists with complaints of severe headache, increase in creatinine to 1.9 worsening hypertension (since 1 month), and subnephrotic proteinuria after 4 years post-transplant (May 2019) with the clinical impression of chronic active antibody-mediated rejection (CABMR), chronic T-cell-mediated renal graft rejection (CATMR), and calcineurin inhibitors (CNI) toxicity. His medical history also revealed Chronic Kidney Disease of unknown origin. He underwent live related (Donor-wife) ABO compatible renal transplant in January 2015. He was kept on triple-drug immunosuppression (prednisolone, MMF, tacrolimus), anti-hypertensive, and maintained baseline creatinine up to 1.2–1.3 mg/dl.

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Renal biopsy revealed only mild mesangioproliferative glomerulonephritis with normal capillary membrane and capillary lumina. Tubules, interstitium, and blood vessels revealed largely unremarkable morphology (Fig. 1a-c). Immunofluorescence study showed full-house pattern of immunofluorescence done with immunoglobulin (Ig)G, IgM, IgA, C3, C1q, Fibrinogen, Kappa, and Lambda (Figs. 2 and 3).

Electron microscopy study showed mild mesangioproliferative glomerulonephropathy with an immune complex of electron-dense deposit but no tubuloreticular bodies (Fig. 4a). C4d by immunohistochemistry study (Fig. 4b) was negative excluding the possibility of CABMR. The absence of tubilitis and interstitial inflammation excluded the possibility of CATMR. Morphological evidence of CNI toxicity was also excluded from the study.

DISCUSSION

Post-transplant glomerulopathy (recurrent or de novo) can be of various morphologies. Glomerulopathy with full-house immunofluorescence pattern suggests the possibility of lupus nephritis and clinical and serological evaluation to exclude the possibility of lupus nephritis which is usually done [5].

After correlation with the American College of Rheumatology (ACR) [1] or Systemic Lupus International Collaborating Clinics (SLICC) [2] classification criteria, a diagnosis of SLE and lupus nephritis is offered. As per these criteria, our case had <4 SLICC and ACR criteria (no clinical symptoms, C3 normal, and antinuclear antibodies (ANA) profile negative) and hence could not be classified

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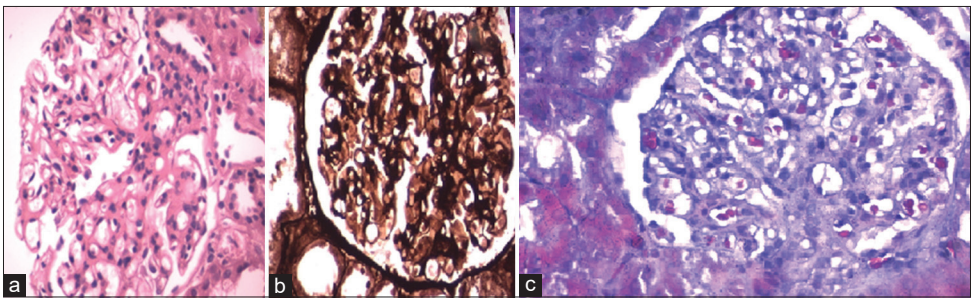

Figure 1: (a) Histopathological examination showed (a) H and E stained section with glomeruli at ×400 shows mild mesangial hypercellularity; (b) Left side of page periodic acid-Schiff (PAS) stained section of glomerulus at ×400 shows mesangial hypercellularity and right side of page PAS stained section with glomerulus on ×400 shows mesangial hypercellularity; (c) Masson's Trichrome-stained section with glomerulus at ×400 shows mesangial hypercellularity

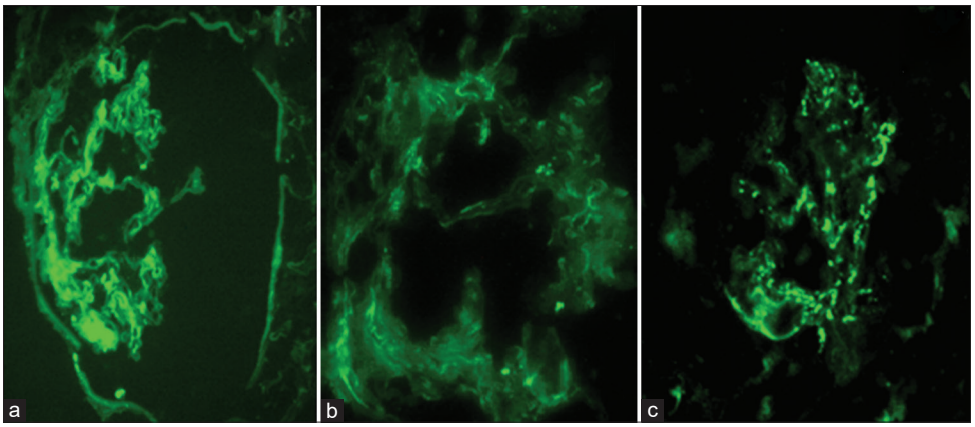

Figure 2: Immunofluorescence study with immunoglobulin (Ig)G, IgM, and IgA from the right to left side of page positive, mesangial, and granular

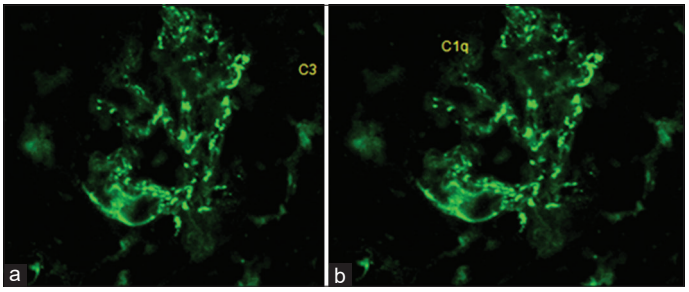

Figure 3: Immunofluorescence study for C3 and C1q show positive, mesangial, and granular

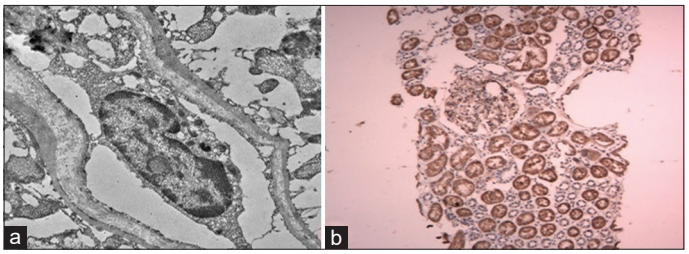

Figure 4: (a) Electronmicroscopy study shows mesangial electron-dense deposit but no tubuloreticular bodies; ×20000; (b) C4d stain by IHC is negative in peritubular capillary wall

as SLE or lupus nephritis, though mesangioproliferative glomerulonephritis is evident that based on the morphology of renal graft biopsy and full-house immunofluorescence study to support other investigators [6,7]. Mesangial cells dense deposits on electron microscopic study supported the diagnosis of transplant glomerulopathy (Fig. 4a). Our case of idiopathic NLFHN had male dominance which is similar to the original article. Normal C3, ANA negative, and <4 SLICC and ACR criteria, as well as mild mesangioproliferative pattern with full-house pattern of immunofluorescence, are observed supporting the presentation of idiopathic non-lupus FHN compared with lupus FH nephropathy (LFHN) male patients [3,7,8]. Electron microscopic study done in our case had electron-dense deposit, but no tubuloreticular body is classical of lupus nephritis.

The patient is followed up for more than 3 years and has not developed any criteria to classify it as systemic lupus nephritis. Even after immunosuppression therapy, there is persistent proteinuria (Table 1) and has become dependent presently. This case, thus, did not fulfill the ACR or SLICC classification criteria anytime during follow-up time too [6,9-11]. However, our case is of post-transplant (4 years), while the original study had all cases in native kidney biopsy (Table 1). Hence, this study case with post-transplant idiopathic NLFHN indicates poor renal survival and, therefore, deserves careful consideration by the nephrologists. Further, such post-transplant NLFHN patients are promptly considered for potential treatment with prognostic clinical evaluation.

Table 1: Comparison of the present case with an original study [6]

S. No.	Original Article	Present Case
1	Native Kidney	4 year post-transplant
2	Majority males	Male
3	ANA profile negative	ANA profile negative
4	C3 normal	C3 normal
5	Predominantly mild mesangioprolifertaive GN and membranous GN	mild mesangioprolifertaive GN
6	<4 SLICC/ACR criteria for SLE	<4 SLICC/ACR criteria for SLE
7	Full-house pattern on IF	Full-house pattern on IF
8	EM one case with no Tubuloreticular body	EM-no Tubuloreticular body
9	With immunosuppression persistence of proteinuria	With increased immunosuppression persistence of proteinuria

SLICC: Systemic lupus international collaborating clinics, ACR: American college of rheumatology

CONCLUSION

To the best of our knowledge, this is the first case of post-transplant NLFHN which is critical to report as these patients usually have poor outcomes. Heavy immunosuppression and cytotoxic drugs are required to achieve remission. Furthermore, treatment protocol needs to be developed for such transplant patients.

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